In horses in Ontario, there is a high prevalence of antibodies to serovar bratislava, for which horses are thought to be a maintenance host (4), but abortions are most commonly associated with serovar pomona. The high titers to 3 common serovars make it difficult to determine on the basis of a single test which serovar(s) was responsible for the infections we report here. We suggest that it was pomona, since this showed the highest titers; the other titers may have been paradoxical reactions that occur early in leptospiral infection. Exposure of mares to serovar pomona could be through direct contact with cattle, swine, rodents, or wildlife, or exposure to urine from these animals; exposure to grippotyphosa might follow direct or indirect encounter with rodents or other wildlife, including raccoons. Infection of dogs with serovar grippotyphosa may be increasing in Ontario, possibly as a result of widespread infection in raccoons (5). In the herds reported here, there was no known exposure to cattle or swine, but pasturing would have provided opportunities for contact with wildlife and urine. The unusually long and mild autumn in eastern Ontario in 1998 may have contributed to increased opportunities for pasture interaction between grazing horses and wildlife.

Diagnosis of this abortion would have been difficult if the placentas had not been submitted, as histological demonstration of leptospires in fetal kidney was inconsistent, and some fetuses had no leptospiral antibodies in body fluids. This report emphasizes the continuing role of leptospires in animal disease, including abortion in horses, and the need for an approved leptospiral vaccine for this species.

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New Brunswick

Infectious salmon anemia in Atlantic salmon

n June 28, 1996, the first author was called to an Atlantic salmon farm near Grand Manan Island in New Brunswick. The site complained of unexplained elevated mortality in one cage of fish. Atlantic salmon weighing approximately 4 kg were examined and found to have pale gills and petechial and ecchymotic hemorrhages in the muscle. Hemorrhage was also observed on the serosa of liver, cecum, spleen, body wall, and other internal organs.

Samples from 4 fish were submitted to the Atlantic Veterinary College for histopathologic examination. One of the fish had lesions consisting of severe sinusoidal congestion and acute multifocal renal tubular necrosis, splenic multifocal and renal interstitial erythrophagocytosis and intravascular hemolysis. There were no lesions of bacterial kidney disease. These findings were consistent with an acute hemolytic event, with either a toxic or an osmoregulatory etiology. The other 3 fish submitted showed reduced erythrocyte numbers, suggesting an ongoing anemic condition.

This cage of fish was killed in 1996, leaving no opportunity for follow-up diagnostic work. The farm also reported having had a cage of fish in 1995 that had experienced similar clinical signs and mortality; these fish were killed in 1995 without diagnostic evaluation.

Around August 1, 1996, the first author identified a second farm, located approximately 50 km from the Grand Manan farm, that had similar clinical signs.

Another veterinarian encountered a similar case on another farm in early August, confirming an emerging serious disease problem. Following extensive diagnostic work, this latter disease was named hemorrhagic kidney syndrome (HKS) (1).

This syndrome was diagnosed on more farms in 1997, resulting in the New Brunswick Department of Food, Fisheries and Aquaculture allocating funding to diagnostic laboratories to investigate its etiology. In 1997, the fish health laboratory of the Research and Productivity Council, Department of Food, Fisheries and Aquaculture isolated the infectious salmon anemia (ISA) virus from tissues of affected fish. This finding was announced in September 1997 (2).

Infectious salmon anemia was first recognized in Norway in 1984 (3). It is characterized by severe anemia and high mortality. Other clinical signs observed in the salmon in Norway included exophthalmia, ascites, congestion and enlargement of the liver and spleen, and petechiation of visceral fat (4). Isolation of the virus was recently made possible by the development of a salmon head kidney (SHK-1) cell line (5). The unavailability of these cell cultures in Canada hampered the initial diagnosis of ISA. Earlier reports from Norway suggested that finding a congested, enlarged, dark brown or black liver was characteristic of ISA. However, this gross lesion was rarely observed in fish in the New Brunswick outbreak (6), a fact which further complicated the diagnosis in 1996 and 1997.

As of November 1998, 22 salmon farms in New Brunswick have been depopulated and other farms are also scheduled for depopulation to try to control the spread of this infection. The Canadian Food Inspection Agency approved an autogenous ISA vaccine for use in fish that will be released into the sea in 1999. Year-class separation of fish and improved biosecurity protocols have been implemented to minimize future impacts of the disease.

The histopathological evidence of ISA in fish from Grand Manan in June 1996 and the subsequent isolation of the virus from affected fish on other New Brunswick Atlantic salmon farms in September 1997 were the first evidence that ISA existed outside Norway. Recently, the disease has been reported in some salmon sites in Scotland (7). Identifying the source of the infection in New Brunswick is complicated by the time that has elapsed since it was first recognized, particularly since it may have existed on the Grand Manan farm as early as 1995.

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